Delayed Medullar Syndrome after Aneurysmal Subarachnoid Haemorrhage
A Case report of Cystic Arachnoiditis!

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Summary

We described a case of chronic spinal cystic arachnoiditis after subarachnoid haemorrhage in a 54-year-old woman with a ruptured vertebral artery aneurysm treated by coils. At three months she complained of lumbar pain. At twelve months she presented lower limbs paresthesia then a rapidly bilateral motor deficit. MR showed a spinal arachnoiditis with two compressive cysts. Surgical decompression was inefficient and after three months spinal compression symptoms worsened and MR signs were unchanged.

Introduction

Chronic spinal cystic arachnoiditis (CSCA) is an uncommon complication of subarachnoid haemorrhage (SAH). Although the incidence of ruptured aneurysms in the general population is about 10/100.000/year, there are only few reports in the literature [1,2,4,6,9-11]. Diagnosis is difficult and often delayed because of very progressive minor symptoms that may be considered functional in the spectrum of complaints patients may present in the months following SAH. We report a case of a CSCA following coiling of a ruptured vertebral aneurysm, which was diagnosed late and only depicted at the time of cord compression.

Case Report

A 54-year-old woman presented with a WFNS Grade I SAH. Head CT scan showed blood predominating at the left cerebellopontine angle cistern and fourth ventricle and a mild ventricular dilatation (figure 1A,B). CT-angiography disclosed a berry aneurysm located at the level of left PICA. The patient deteriorated rapidly (Grade IV WFNS). CT scan showed worsened ventricular dilatation with no sign of rebleeding. External ventricular drainage was placed. Cerebral angiography confirmed the aneurysm of the left intradural vertebral artery (6.7 mm large x 3.3 mm depth, and 3 mm neck), located 2 cm proximal to the origin of PICA (figure 2A). Embolization was done with the balloon remodelling technique introducing two GDC 10 coils (Boston Scientific, Fremont, CA) (figure 2B). About 48h after embolization, she had a slight right lower limb paresis and diplopia without other neurological symptoms. During the following days she presented an infectious respiratory syndrome managed by tracheotomy and antibiotics. One month after her admission she was discharged home with still a slight diplopia and right lower limb paresis.

At three months, she complained of lumbar pain. Brain MR and MRA showed no parenchymal lesion, no ventricular dilatation and persistent good aneurysm occlusion. Twelve months after the SAH, she complained of progressively increasing paresthesia of both lower limbs. Clinical examination was normal. Ventricle size was normal on CT Scan. In the next weeks, she suffered gait disturbances, then slight urinary dysfunction. These symptoms worsened and fifteen months after SAH, she had a pyramidal syndrome with bilateral motor
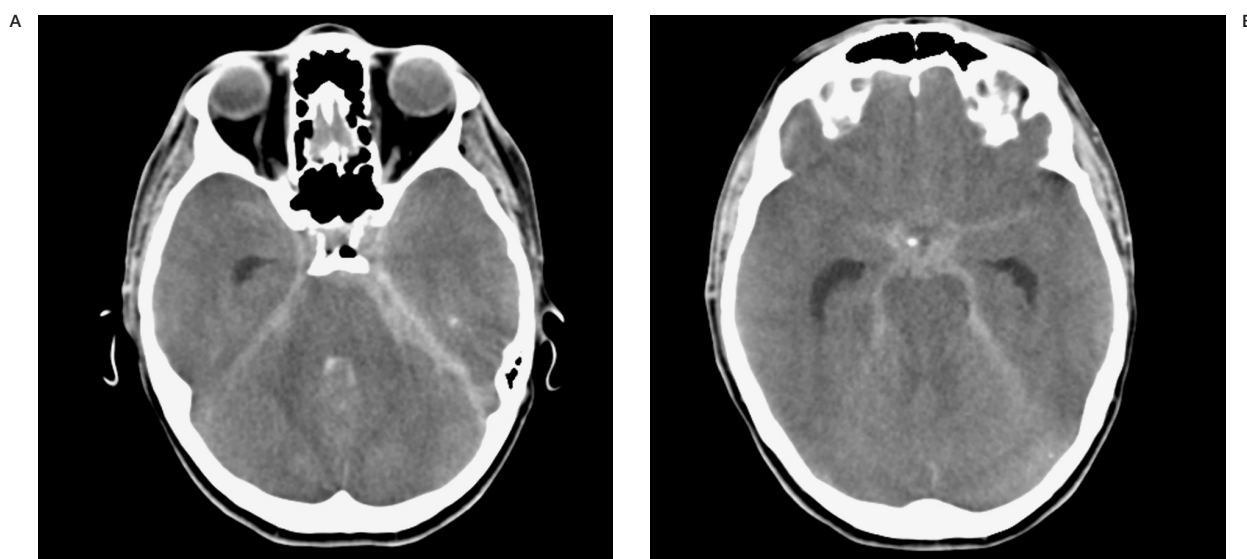
Figure 1 A,B) Initial Non-contrast CT scan shows a SAH predominating at the left cerebellopontine angle cistern and fourth ventricle and a mild ventricular dilatation.
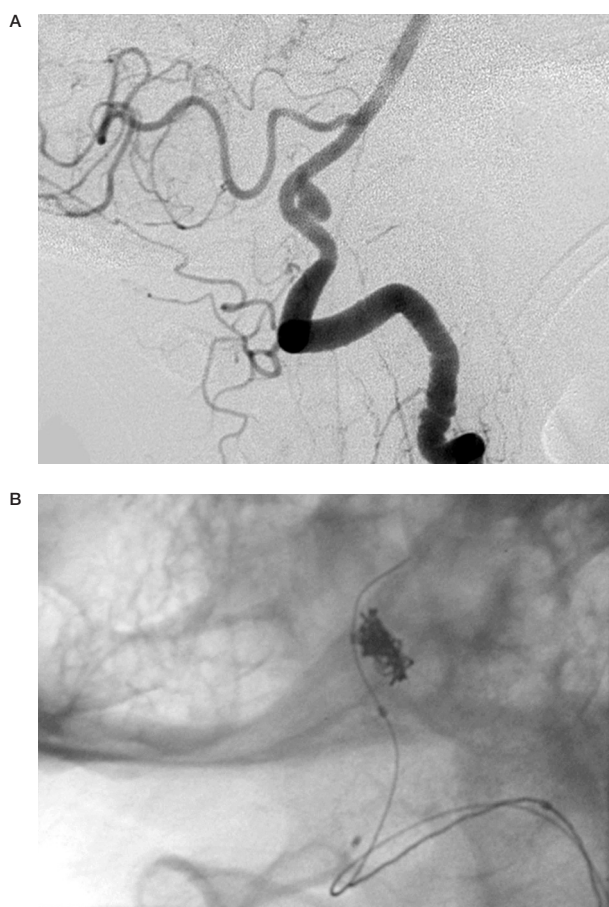

Figure 2 A,B) Cerebral angiography shows a left intradural vertebral artery aneurysm located 2 cm proximal to the origin of PICA (A). Coil mesh after embolization (B).
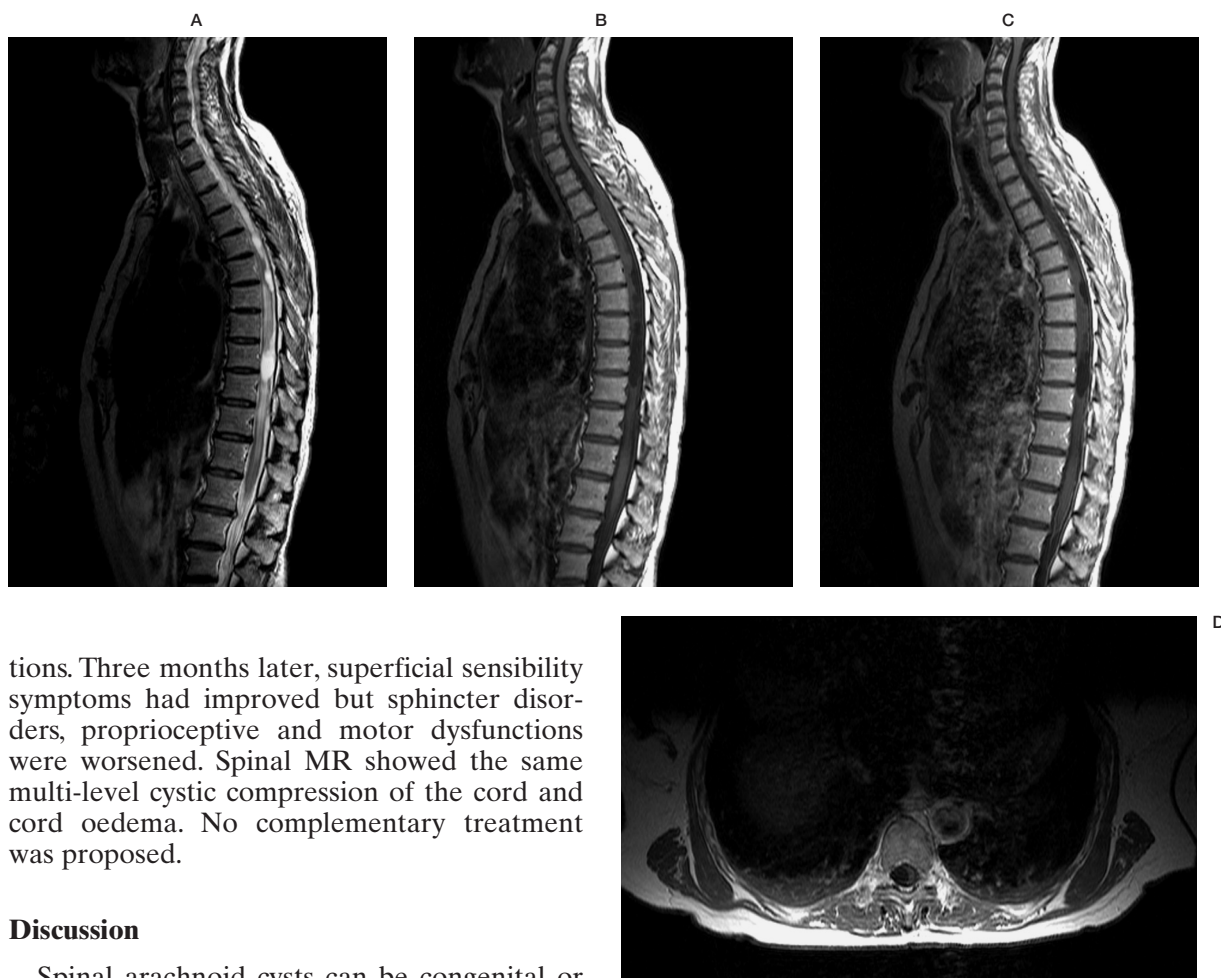
deficit, sensitive proprioceptive symptoms, and superficial sensibility deficit with a hypoesthesia until the level of T10. Somesthesic and motor evoked potentials were abnormal.

Brain MR was normal (not shown). Spinal MR (figure 3 A-D) showed intradural extramedullary well defined cystic lesions, markedly hypointense on T1 and hyperintense on T2 sequences, extending from the level of T3 to T10. Two cysts were producing a mass effect on the spinal cord major at T9/T10 and minor at T6/T7. Oedema of the spinal cord under the level of T6 until the conus medullaris was visible. There was a discrete meningeal Gadolinium enhancement of the conus medullaris but no enhancement of cystic lesions.

Considering the history of SAH, clinical symptoms and MR findings the diagnosis of spinal arachnoid cysts due to chronic arachnoiditis secondary to SAH was proposed.

Because of the rapidly worsening motor deficit, walking being difficult even with a stick, surgery was decided. The goal was to decompress the spinal cord at the level of the T9-T10 cyst. After a T8-T9-T10 laminectomy and opening of the dura mater, the arachnoid membrane appeared abnormally white and extremely thick. It was open on 3 cm at the level of the cyst that was located on the posterior and right side aspect of the cord. No shunt was placed.

The patient was discharged at home six days after surgery without post surgical complica-

Figure 3 A-D) Thoraco-lumbar MR on sagittal T2 (A), T1 (B), T1 post-Gadolinium (C) and axial T1 (D), show intradural extramedullary well defined cystic lesions extending from T3 to T10. Oedema of the spinal cord is visible under the level of T6 until the conus medullaris. Note the discrete gadolinium enhancement of the surface of the conus.

tions. Three months later, superficial sensibility symptoms had improved but sphincter disorders, proprioceptive and motor dysfunctions were worsened. Spinal MR showed the same multi-level cystic compression of the cord and cord oedema. No complementary treatment was proposed.

Discussion

Spinal arachnoid cysts can be congenital or acquired. The acquired cysts have many origins such as: spinal cord trauma or surgery, infections and other injuries that cause inflammation and subarachnoid adhesions[12]. The origin of arachnoid cysts after SAH is not completely understood.

The products of blood cells degradation during haemolysis can produce an inflammatory reaction of arachnoid membranes that can lead to fibrosis[11]. A few papers reported an increased concentration of procollagen peptides in the CSF after SAH, thus supporting a fibroproliferative reaction and fibrosis[7,8]. The scar tissue can cause septation of the subarachnoid space producing pockets of CSF that may be loculated within the arachnoid membranes. Continuous production of CSF and filling of these cysts without enough evacuation may induce compression of the spinal cord[11].

In addition, manipulation of the subarachnoid space by ventricular shunting, cisternotomy, or repeated lumbar punctures may favour such inflammatory reaction. In the literature

some cases have occurred after manipulations of the spinal subarachnoid space[9,11], others occurred without any manipulation, the blood itself being probably the only source of the inflammation[4,6]. In our case, there was no manipulation of the spinal subarachnoid space. The only surgical procedure consisted in an external ventricular shunt that was removed as soon as hydrocephalus resolved. To our knowledge this is the first report of a spinal cystic arachnoiditis developing after SAH due to a posterior fossa aneurysm treated by coils. Posterior location of the aneurysm may favour the occurrence of arachnoiditis. Nevertheless, in the review by Kok et Al[4], only six of fourteen cases reported concerned posterior artery aneur-

ysms. They raised the hypothesis that such aneurysms produce more blood in the spinal subarachnoid space.

We can postulate that the amount of blood after SAH play a role in the pathogenesis of spinal arachnoid cysts. In the case of Tumialan et Al [11] and the two cases of Kok et Al [4], the SAHs were Grade IV in Fisher classification with intraventricular blood. Besides, because of bed rest, the blood tends to accumulate in the thoracic spinal subarachnoid space [4,11]. The duration of bed rest after SAH could then be an associated factor favouring arachnoiditis.

The first complaint in our patient was a chronic lumbar pain which lasted for months before symptoms of cord compression. In the literature, reported symptoms are variable and include lower limb dysesthesia and pain, progressive paraparesis and sphincter disturbances, sometimes combined with sensory loss [12]. Chronic cystic arachnoiditis is a rarely observed complication post-SAH. It is probably underestimated and lumbar pains are frequent symptoms after SAH that may be due in some cases to minimal arachnoiditis. In addition, lower limb weakness and gait disturbances are often attributed to hydrocephalus and the spine is rarely incriminated. In our patient, the lumbar pain occurred at three months, paresthesia of both lower limbs at twelve months but diagnosis was only made at fourteen months when the patient presented obvious cord compression with bilateral motor deficit rapidly worsening. It required surgery when most cases published were treated conservatively.

Surgical treatment of such lesions therefore has been rarely described and consisted in decompression by total excision when possible, fenestration or placement of a shunt. Surgical results depend on age, duration of paresis and degree of cord damage [3,5]. In our case, surgery was difficult due to extreme thickening of the arachnoid membrane and extension of the arachnoiditis from T3 to T10. It consisted in opening of the membrane at the level of the most compressive cyst on 3 cm. Surgery was finally inefficient and three months after operation sphincter disorders, proprioceptive and motor dysfunctions were worsened. Spinal MR showed the same multilevel cystic compression of the cord and cord oedema. It is difficult to determine if an earlier diagnosis and surgery would have avoided such motor deficit and make surgery easier and more efficient.

Conclusions

CSCA are rare but potentially devastating complications of SAH. Their origin and reason for rarity are still poorly understood. Posterior circulation aneurysms, a huge amount of blood and long duration of bed rest may increase the risk of arachnoiditis. Surgical spinal manipulation may be an associated factor. The diagnosis is probably underestimated in cases of patients presenting with only lumbar pain and lower limbs paresthesia.

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